09/419,901
L4/Cook 9/5/06
updated Search

d his

(FILE 'HOME' ENTERED AT 10:53:41 ON 05 SEP 2006)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, JAPIO' ENTERED AT 10:54:00 ON 05 SEP 2006

L1	150	S (CHEMICAL ADDUCT)
L2	58	S L1 AND DAMAGE?
L3	25	DUPLICATE REMOVE L2 (33 DUPLICATES REMOVED)
L4	1	S L3 AND TROPONIN?
L5	1	S L3 AND MYOSIN?
L6	0	S L3 AND ACTININ?
L7	24	S L3 NOT L4
L8	1	S L1 AND MUSCLE?
L9	1	S L1 AND TROPONIN?
L10	1	S L1 AND MYOSIN?
L11	15	S L1 AND REVIEW?
L12	11	DUPLICATE REMOVE L11 (4 DUPLICATES REMOVED)
L13	39	S TROPONIN AND ADDUCT
L14	16	DUPLICATE REMOVE L13 (23 DUPLICATES REMOVED)
L15	6	S L14 AND MUSCLE?

d his

(FILE 'HOME' ENTERED AT 10:53:41 ON 05 SEP 2006)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, JAPIO' ENTERED AT 10:54:00 ON 05 SEP 2006

L1	150	S (CHEMICAL ADDUCT)
L2	58	S L1 AND DAMAGE?
L3	25	DUPLICATE REMOVE L2 (33 DUPLICATES REMOVED)
L4	1	S L3 AND TROPONIN?
L5	1	S L3 AND MYOSIN?
L6	0	S L3 AND ACTININ?
L7	24	S L3 NOT L4
L8	1	S L1 AND MUSCLE?
L9	1	S L1 AND TROPONIN?
L10	1	S L1 AND MYOSIN?
L11	15	S L1 AND REVIEW?
L12	11	DUPLICATE REMOVE L11 (4 DUPLICATES REMOVED)
L13	39	S TROPONIN AND ADDUCT
L14	16	DUPLICATE REMOVE L13 (23 DUPLICATES REMOVED)
L15	6	S L14 AND MUSCLE?

ANSWER 2 OF 6 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 1996:109721 BIOSIS
DN PREV199698681856
TI Covalent binding of peptides to the N-terminal hydrophobic region of cardiac troponin C has limited effects on function.
AU Lin, Xin; Dotson, Darrell G.; Putkey, John A. [Reprint author]
CS Dep. Biochem. Mol. Biol., Univ. Tex. Med. Sch., 6431 Fannin St., Houston, TX 77030, USA
SO Journal of Biological Chemistry, (1996) Vol. 271, No. 1, pp. 244-249. CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 12 Mar 1996
Last Updated on STN: 13 Mar 1996
AB Exposure of an N-terminal hydrophobic region in troponin C is thought to be important for the regulation of contraction in striated muscle. To test this hypothesis, single Cys residues were engineered at positions 45, 81, 84, or 85 in the N-terminal hydrophobic region of cardiac troponin C (cTnC) to provide specific sites for attachment of blocking groups. A synthetic peptide, AcVal-Arg-Ala-Ile-Gly-Lys-Leu-Ser-Ser, or biotin was coupled to these Cys residues, and the covalent adducts were tested for activity in cTnC-extracted myofibrils. Covalent modification of cTnC(C45) had no effect on maximal myofibril ATPase activity. Greatly decreased myofibril ATPase activity (70-80% inhibited) resulted when the peptide was conjugated to Cys-81 in cTnC(C81), while a lesser degree of inhibition (10-25% inhibited) resulted from covalent modification of cTnC(C84) and cTnC(C85). Inhibition was not due to an altered affinity of the cTnC(C81)/peptide conjugate for the myofibrils, and the Ca-2+ dependence of ATPase activity was essentially identical to the unmodified protein. Thus, a subregion of the N-terminal hydrophobic region in cTnC is sensitive to disruption, while other regions are less important or can adapt to rather bulky blocking groups. The data suggest that Ca-2+--sensitizing drugs may bind to the N-terminal hydrophobic region on cTnC but not interfere with transmission of the Ca-2+ signal.
CC Cytology - Animal 02506
Biochemistry studies - Nucleic acids, purines and pyrimidines 10062
Biochemistry studies - Proteins, peptides and amino acids 10064
Biophysics - Molecular properties and macromolecules 10506
Enzymes - Chemical and physical 10806
Cardiovascular system - Physiology and biochemistry 14504
Muscle - Physiology and biochemistry 17504
IT Major Concepts
Biochemistry and Molecular Biophysics; Cardiovascular System (Transport and Circulation); Cell Biology; Enzymology (Biochemistry and Molecular Biophysics); Muscular System (Movement and Support)
IT Chemicals & Biochemicals
CALCIUM ION; ATPASE
IT Miscellaneous Descriptors
ATPASE ACTIVITY; CALCIUM ION SIGNAL TRANSMISSION; STRIATED MUSCLE
ORGN Classifier
Animalia 33000
Super Taxa
Animalia
Organism Name
Animalia
Taxa Notes
Animals
RN 14127-61-8 (CALCIUM ION)
9000-83-3 (ATPASE)

ANSWER 2 OF 6 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 1996:109721 BIOSIS
DN PREV199698681856
TI Covalent binding of peptides to the N-terminal hydrophobic region of cardiac troponin C has limited effects on function.
AU Lin, Xin; Dotson, Darrell G.; Putkey, John A. [Reprint author]
CS Dep. Biochem. Mol. Biol., Univ. Tex. Med. Sch., 6431 Fannin St., Houston, TX 77030, USA
SO Journal of Biological Chemistry, (1996) Vol. 271, No. 1, pp. 244-249. CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 12 Mar 1996
Last Updated on STN: 13 Mar 1996
AB Exposure of an N-terminal hydrophobic region in troponin C is thought to be important for the regulation of contraction in striated muscle. To test this hypothesis, single Cys residues were engineered at positions 45, 81, 84, or 85 in the N-terminal hydrophobic region of cardiac troponin C (cTnC) to provide specific sites for attachment of blocking groups. A synthetic peptide, AcVal-Arg-Ala-Ile-Gly-Lys-Leu-Ser-Ser, or biotin was coupled to these Cys residues, and the covalent adducts were tested for activity in TnC-extracted myofibrils. Covalent modification of cTnC(C45) had no effect on maximal myofibril ATPase activity. Greatly decreased myofibril ATPase activity (70-80% inhibited) resulted when the peptide was conjugated to Cys-81 in cTnC(C81), while a lesser degree of inhibition (10-25% inhibited) resulted from covalent modification of cTnC(C84) and cTnC(C85). Inhibition was not due to an altered affinity of the cTnC(C81)/peptide conjugate for the myofibrils, and the Ca-2+ dependence of ATPase activity was essentially identical to the unmodified protein. Thus, a subregion of the N-terminal hydrophobic region in cTnC is sensitive to disruption, while other regions are less important or can adapt to rather bulky blocking groups. The data suggest that Ca-2+--sensitizing drugs may bind to the N-terminal hydrophobic region on cTnC but not interfere with transmission of the Ca-2+ signal.
CC Cytology - Animal 02506
Biochemistry studies - Nucleic acids, purines and pyrimidines 10062
Biochemistry studies - Proteins, peptides and amino acids 10064
Biophysics - Molecular properties and macromolecules 10506
Enzymes - Chemical and physical 10806
Cardiovascular system - Physiology and biochemistry 14504
Muscle - Physiology and biochemistry 17504
IT Major Concepts
Biochemistry and Molecular Biophysics; Cardiovascular System (Transport and Circulation); Cell Biology; Enzymology (Biochemistry and Molecular Biophysics); Muscular System (Movement and Support)
IT Chemicals & Biochemicals
CALCIUM ION; ATPASE
IT Miscellaneous Descriptors
ATPASE ACTIVITY; CALCIUM ION SIGNAL TRANSMISSION; STRIATED MUSCLE
ORGN Classifier
Animalia 33000
Super Taxa
Animalia
Organism Name
Animalia
Taxa Notes
Animals
RN 14127-61-8 (CALCIUM ION)
9000-83-3 (ATPASE)

ANSWER 5 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN
AN 1996:36916 CAPLUS
DN 124:173661
ED Entered STN: 18 Jan 1996
TI Toxic plant residues in meat
AU Seawright, A. A.
CS Natl. Res. Cent. Environmental Toxicol., Coopers Plains, QLD 4108, Australia
SO Plant-Associated Toxins: Agricultural, Phytochemical and Ecological Aspects, [Proceedings of the International Symposium on Poisonous Plants], 4th, Fremantle, Australia, Sept. 26-Oct. 1, 1993 (1994), Meeting Date 1993, 77-82. Editor(s): Colegate, Steven M.; Dorling, Peter R. Publisher: CAB International, Wallingford, UK.
CODEN: 62FFAS
DT Conference; General Review
LA English
CC 17-0 (Food and Feed Chemistry)
AB A review with 26 refs. which discusses the possible health significance of chemical residues in the meat of food animals. The review discusses three issues: 1- the possibility of secondary poisoning, 2- the accumulation of potentially injurious unmetabolized chems. in the tissues of apparently normal animals and 3- the relationship of the presence of chem. adduct residues in tissues to the initiation of progressive diseases.
ST review meat contamination plant toxin
IT Food contamination
Meat
(toxic plant residues in meat)

ANSWER 5 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN
AN 1996:36916 CAPLUS
DN 124:173661
ED Entered STN: 18 Jan 1996
TI Toxic plant residues in meat
AU Seawright, A. A.
CS Natl. Res. Cent. Environmental Toxicol., Coopers Plains, QLD 4108, Australia
SO Plant-Associated Toxins: Agricultural, Phytochemical and Ecological Aspects, [Proceedings of the International Symposium on Poisonous Plants], 4th, Fremantle, Australia, Sept. 26-Oct. 1, 1993 (1994), Meeting Date 1993, 77-82. Editor(s): Colegate, Steven M.; Dorling, Peter R. Publisher: CAB International, Wallingford, UK.
CODEN: 62FFAS
DT Conference; General Review
LA English
CC 17-0 (Food and Feed Chemistry)
AB A review with 26 refs. which discusses the possible health significance of chemical residues in the meat of food animals. The review discusses three issues: 1- the possibility of secondary poisoning, 2- the accumulation of potentially injurious unmetabolized chems. in the tissues of apparently normal animals and 3- the relationship of the presence of chem. adduct residues in tissues to the initiation of progressive diseases.
ST review meat contamination plant toxin
IT Food contamination
Meat
(toxic plant residues in meat)

ANSWER 3 OF 6 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 1996:106227 BIOSIS
DN PREV199698678362
TI Isolation, characterization, and partial purification of a novel ubiquitin-protein ligase, E3: Targeting of protein substrates via multiple and distinct recognition signals and conjugating enzymes.
AU Gonen, Hedva; Stancovski, Ilana; Shkedy, Dganit; Hadari, Tamar; Bercovich, Beatrice; Bengal, Eyal; Mesilati, Shlomit; Abu-Hatou, Ossama; Schwartz, Alan L.; Ciechanover, Aaron [Reprint author]
CS Dep. Biochem., Fac. Medicine, Technion-Israel Inst. Technol., P.O. Box 9649, Haifa 31096, Israel
SO Journal of Biological Chemistry, (1996) Vol. 271, No. 1, pp. 302-310. CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 12 Mar 1996
Last Updated on STN: 13 Mar 1996
AB Degradation of a protein via the ubiquitin system involves two discrete steps, conjugation of ubiquitin to the substrate and degradation of the adduct. Conjugation follows a three-step mechanism. First, ubiquitin is activated by the ubiquitin-activating enzyme, E1. Following activation, one of several E2 enzymes (ubiquitin-carrier proteins or ubiquitin-conjugating enzymes, UBCs) transfers ubiquitin from E1 to the protein substrate that is bound to one of several ubiquitin-protein ligases, E3s. These enzymes catalyze the last step in the process, covalent attachment of ubiquitin to the protein substrate. The binding of the substrate to E3 is specific and implies that E3s play a major role in recognition and selection of proteins for conjugation and subsequent degradation. So far, only a few ligases have been identified, and it is clear that many more have not been discovered yet. Here, we describe a novel ligase that is involved in the conjugation and degradation of non "N-end rule" protein substrates such as actin, troponin T, and MyoD. This substrate specificity suggests that the enzyme may be involved in degradation of muscle proteins. The ligase acts in concert with E2-F1, a previously described non N-end rule UBC. Interestingly, it is also involved in targeting lysozyme, a bona fide N-end rule substrate that is recognized by E3-alpha and E2-14 kDa. The novel ligase recognizes lysozyme via a signal(s) that is distinct from the N-terminal residue of the protein. Thus, it appears that certain proteins can be targeted via multiple recognition motifs and distinct pairs of conjugating enzymes. We have purified the ligase apprx 200-fold and demonstrated that it is different from other known E3s, including E3-alpha/UBR1, E3-beta, and E6-AP. The native enzyme has an apparent molecular mass of apprx 550 kDa and appears to be a homodimer. Because of its unusual size, we designated this novel ligase E3L (large). E3L contains an -SH group that is essential for its activity. Like several recently described E3 enzymes, including E6-AP and the ligase involved in the processing of p105, the NF-kappa-B precursor, the novel ligase is found in mammalian tissues but not in wheat germ.
CC Cytology - Animal 02506
Biochemistry studies - Proteins, peptides and amino acids 10064
Enzymes - Chemical and physical 10806
Metabolism - Proteins, peptides and amino acids 13012
Blood - Blood cell studies 15004
Blood - Lymphatic tissue and reticuloendothelial system 15008
Muscle - Physiology and biochemistry 17504
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Cell Biology; Enzymology (Biochemistry and Molecular Biophysics); Metabolism; Muscular System (Movement and Support)
IT Chemicals & Biochemicals
UBIQUITIN; LIGASE
IT Miscellaneous Descriptors
MUSCLE; RETICULOCYTE

ANSWER 3 OF 6 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 1996:106227 BIOSIS
DN PREV199698678362
TI Isolation, characterization, and partial purification of a novel ubiquitin-protein ligase, E3: Targeting of protein substrates via multiple and distinct recognition signals and conjugating enzymes.
AU Gonen, Hedva; Stancovski, Ilana; Shkedy, Dganit; Hadari, Tamar; Bercovich, Beatrice; Bengal, Eyal; Mesilati, Shlomit; Abu-Hatou, Ossama; Schwartz, Alan L.; Ciechanover, Aaron [Reprint author]
CS Dep. Biochem., Fac. Medicine, Technion-Israel Inst. Technol., P.O. Box 9649, Haifa 31096, Israel
SO Journal of Biological Chemistry, (1996) Vol. 271, No. 1, pp. 302-310. CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 12 Mar 1996
Last Updated on STN: 13 Mar 1996
AB Degradation of a protein via the ubiquitin system involves two discrete steps, conjugation of ubiquitin to the substrate and degradation of the adduct. Conjugation follows a three-step mechanism. First, ubiquitin is activated by the ubiquitin-activating enzyme, E1. Following activation, one of several E2 enzymes (ubiquitin-carrier proteins or ubiquitin-conjugating enzymes, UBCs) transfers ubiquitin from E1 to the protein substrate that is bound to one of several ubiquitin-protein ligases, E3s. These enzymes catalyze the last step in the process, covalent attachment of ubiquitin to the protein substrate. The binding of the substrate to E3 is specific and implies that E3s play a major role in recognition and selection of proteins for conjugation and subsequent degradation. So far, only a few ligases have been identified, and it is clear that many more have not been discovered yet. Here, we describe a novel ligase that is involved in the conjugation and degradation of non "N-end rule" protein substrates such as actin, troponin T, and MyoD. This substrate specificity suggests that the enzyme may be involved in degradation of muscle proteins. The ligase acts in concert with E2-F1, a previously described non N-end rule UBC. Interestingly, it is also involved in targeting lysozyme, a bona fide N-end rule substrate that is recognized by E3-alpha and E2-14 kDa. The novel ligase recognizes lysozyme via a signal(s) that is distinct from the N-terminal residue of the protein. Thus, it appears that certain proteins can be targeted via multiple recognition motifs and distinct pairs of conjugating enzymes. We have purified the ligase apprx 200-fold and demonstrated that it is different from other known E3s, including E3-alpha/UBR1, E3-beta, and E6-AP. The native enzyme has an apparent molecular mass of apprx 550 kDa and appears to be a homodimer. Because of its unusual size, we designated this novel ligase E3L (large). E3L contains an -SH group that is essential for its activity. Like several recently described E3 enzymes, including E6-AP and the ligase involved in the processing of p105, the NF-kappa-B precursor, the novel ligase is found in mammalian tissues but not in wheat germ.
CC Cytology - Animal 02506
Biochemistry studies - Proteins, peptides and amino acids 10064
Enzymes - Chemical and physical 10806
Metabolism - Proteins, peptides and amino acids 13012
Blood - Blood cell studies 15004
Blood - Lymphatic tissue and reticuloendothelial system 15008
Muscle - Physiology and biochemistry 17504
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Cell Biology; Enzymology (Biochemistry and Molecular Biophysics); Metabolism; Muscular System (Movement and Support)
IT Chemicals & Biochemicals
UBIQUITIN; LIGASE
IT Miscellaneous Descriptors
MUSCLE; RETICULOCYTE

ANSWER 2 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

AN 1996:106227 BIOSIS

DN PREV199698678362

TI Isolation, characterization, and partial purification of a novel ubiquitin-protein ligase, E3: Targeting of protein substrates via multiple and distinct recognition signals and conjugating enzymes.

AU Gonen, Hedva; Stancovski, Ilana; Shkedy, Dganit; Hadari, Tamar; Bercovich, Beatrice; Bengal, Eyal; Mesilati, Shlomit; Abu-Hatou, Ossama; Schwartz, Alan L.; Ciechanover, Aaron [Reprint author]

CS Dep. Biochem., Fac. Medicine, Technion-Israel Inst. Technol., P.O. Box 9649, Haifa 31096, Israel

SO Journal of Biological Chemistry, (1996) Vol. 271, No. 1, pp. 302-310.
CODEN: JBCHA3. ISSN: 0021-9258.

DT Article

LA English

ED Entered STN: 12 Mar 1996
Last Updated on STN: 13 Mar 1996

AB Degradation of a protein via the ubiquitin system involves two discrete steps, conjugation of ubiquitin to the substrate and degradation of the adduct. Conjugation follows a three-step mechanism. First, ubiquitin is activated by the ubiquitin-activating enzyme, E1. Following activation, one of several E2 enzymes (ubiquitin-carrier proteins or ubiquitin-conjugating enzymes, UBCs) transfers ubiquitin from E1 to the protein substrate that is bound to one of several ubiquitin-protein ligases, E3s. These enzymes catalyze the last step in the process, covalent attachment of ubiquitin to the protein substrate. The binding of the substrate to E3 is specific and implies that E3s play a major role in recognition and selection of proteins for conjugation and subsequent degradation. So far, only a few ligases have been identified, and it is clear that many more have not been discovered yet. Here, we describe a novel ligase that is involved in the conjugation and degradation of non "N-end rule" protein substrates such as actin, troponin T, and MyoD. This substrate specificity suggests that the enzyme may be involved in degradation of muscle proteins. The ligase acts in concert with E2-F1, a previously described non N-end rule UBC. Interestingly, it is also involved in targeting lysozyme, a bona fide N-end rule substrate that is recognized by E3-alpha and E2-14 kDa. The novel ligase recognizes lysozyme via a signal(s) that is distinct from the N-terminal residue of the protein. Thus, it appears that certain proteins can be targeted via multiple recognition motifs and distinct pairs of conjugating enzymes. We have purified the ligase apprx 200-fold and demonstrated that it is different from other known E3s, including E3-alpha/UBR1, E3-beta, and E6-AP. The native enzyme has an apparent molecular mass of apprx 550 kDa and appears to be a homodimer. Because of its unusual size, we designated this novel ligase E3L (large). E3L contains an -SH group that is essential for its activity. Like several recently described E3 enzymes, including E6-AP and the ligase involved in the processing of p105, the NF-kappa-B precursor, the novel ligase is found in mammalian tissues but not in wheat germ.

CC Cytology - Animal 02506
Biochemistry studies - Proteins, peptides and amino acids 10064
Enzymes - Chemical and physical 10806
Metabolism - Proteins, peptides and amino acids 13012
Blood - Blood cell studies 15004
Blood - Lymphatic tissue and reticuloendothelial system 15008
Muscle - Physiology and biochemistry 17504

IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Cell Biology; Enzymology (Biochemistry and Molecular Biophysics); Metabolism; Muscular System (Movement and Support)

IT Chemicals & Biochemicals
UBIQUITIN; LIGASE

IT Miscellaneous Descriptors

ANSWER 2 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

AN 1996:106227 BIOSIS

DN PREV199698678362

TI Isolation, characterization, and partial purification of a novel ubiquitin-protein ligase, E3: Targeting of protein substrates via multiple and distinct recognition signals and conjugating enzymes.

AU Gonen, Hedva; Stancovski, Ilana; Shkedy, Dganit; Hadari, Tamar; Bercovich, Beatrice; Bengal, Eyal; Mesilati, Shlomit; Abu-Hatou, Ossama; Schwartz, Alan L.; Ciechanover, Aaron [Reprint author]

CS Dep. Biochem., Fac. Medicine, Technion-Israel Inst. Technol., P.O. Box 9649, Haifa 31096, Israel

SO Journal of Biological Chemistry, (1996) Vol. 271, No. 1, pp. 302-310.
CODEN: JBCHA3. ISSN: 0021-9258.

DT Article

LA English

ED Entered STN: 12 Mar 1996
Last Updated on STN: 13 Mar 1996

AB Degradation of a protein via the ubiquitin system involves two discrete steps, conjugation of ubiquitin to the substrate and degradation of the adduct. Conjugation follows a three-step mechanism. First, ubiquitin is activated by the ubiquitin-activating enzyme, E1. Following activation, one of several E2 enzymes (ubiquitin-carrier proteins or ubiquitin-conjugating enzymes, UBCs) transfers ubiquitin from E1 to the protein substrate that is bound to one of several ubiquitin-protein ligases, E3s. These enzymes catalyze the last step in the process, covalent attachment of ubiquitin to the protein substrate. The binding of the substrate to E3 is specific and implies that E3s play a major role in recognition and selection of proteins for conjugation and subsequent degradation. So far, only a few ligases have been identified, and it is clear that many more have not been discovered yet. Here, we describe a novel ligase that is involved in the conjugation and degradation of non "N-end rule" protein substrates such as actin, troponin T, and MyoD. This substrate specificity suggests that the enzyme may be involved in degradation of muscle proteins. The ligase acts in concert with E2-F1, a previously described non N-end rule UBC. Interestingly, it is also involved in targeting lysozyme, a bona fide N-end rule substrate that is recognized by E3-alpha and E2-14 kDa. The novel ligase recognizes lysozyme via a signal(s) that is distinct from the N-terminal residue of the protein. Thus, it appears that certain proteins can be targeted via multiple recognition motifs and distinct pairs of conjugating enzymes. We have purified the ligase apprx 200-fold and demonstrated that it is different from other known E3s, including E3-alpha/UBR1, E3-beta, and E6-AP. The native enzyme has an apparent molecular mass of apprx 550 kDa and appears to be a homodimer. Because of its unusual size, we designated this novel ligase E3L (large). E3L contains an -SH group that is essential for its activity. Like several recently described E3 enzymes, including E6-AP and the ligase involved in the processing of p105, the NF-kappa-B precursor, the novel ligase is found in mammalian tissues but not in wheat germ.

CC Cytology - Animal 02506
Biochemistry studies - Proteins, peptides and amino acids 10064
Enzymes - Chemical and physical 10806
Metabolism - Proteins, peptides and amino acids 13012
Blood - Blood cell studies 15004
Blood - Lymphatic tissue and reticuloendothelial system 15008
Muscle - Physiology and biochemistry 17504

IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Cell Biology; Enzymology (Biochemistry and Molecular Biophysics); Metabolism; Muscular System (Movement and Support)

IT Chemicals & Biochemicals
UBIQUITIN; LIGASE

IT Miscellaneous Descriptors

MUSCLE; RETICULOCYTE
ORGN Classifier
Leporidae 86040
Super Taxa
Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
rabbit
Taxa Notes
Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates
RN 60267-61-0 (UBIQUITIN)
9031-56-5 (LIGASE)
9080-13-1Q (LIGASE)

MUSCLE; RETICULOCYTE
ORGN Classifier
Leporidae 86040
Super Taxa
Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
rabbit
Taxa Notes
Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates
RN 60267-61-0 (UBIQUITIN)
9031-56-5 (LIGASE)
9080-13-1Q (LIGASE)

ANSWER 3 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

AN 1994:212708 BIOSIS

DN PREV199497225708

TI Tropomyosin inhibits the glutaraldehyde-induced cross-link between the central 48-kDa fragment of myosin head and segment 48-67 inactin subdomain 2.

AU Bonafe, Nathalie; Mathieu, Magali; Kassab, Ridha; Chaussepied, Patrick [Reprint author]

CS Centre de Recherches de Biochemie Macromoleculaire, Route de Mende, BP 5051, 34033 Montpellier Cedex, France

SO Biochemistry, (1994) Vol. 33, No. 9, pp. 2594-2603.
CODEN: BICHAW. ISSN: 0006-2960.

DT Article

LA English

ED Entered STN: 10 May 1994
Last Updated on STN: 25 Jun 1994

AB The glutaraldehyde-induced cross-linking of the F-actin-myosin head (S1) complex, previously described (Bertrand et al. (1988) Biochemistry 27, 5728-5736), was investigated in the presence of tropomyosin (Tm) alone or associated with troponin (Tn), at a Tm-Tn/actin/S1 molar ratio of 1:7:3. Among the two acto-S1 cross-linked products with apparent masses of 165 and 200 kDa generated in the absence of the regulatory proteins, only the 165-kDa adduct was formed in the presence of Tm. An identical result was obtained with and without Tn regardless of the presence of Ca-2+ and/or Mg-2+-ADP. The abolition of the 200-kDa cross-linked acto-S1 species was independent of the S1/actin ratio since even a 3-fold excess of S1 over actin, sufficient for fully turning on the thin filament, could not restore the 200-kDa covalent involved in the Ca-2+-linked regulation of the acto-S1 ATPase activity, as the enzymatic activities of both types of complexes were regulated to the same extent by Ca-2+/EGTA, in the presence of the regulatory proteins. Cross-linking experiments performed with (14C) glutaraldehyde showed that both covalent complexes were composed of 1 mol of actin bound to 1 mol of S1 heavy chain. The use of proteolytic actin or S1 derivatives together with the direct proteolysis of the acto-S1 covalent adducts revealed that Tm abolished the cross-link between the central 48-kDa fragment of the S1 heavy chain and Lys-50 of actin subdomain 2 that is responsible for the formation of the 200-kDa entity, while it did not affect the cross-link between the S1 heavy chain segment of residues 636-642 and Arg-28 of actin that generates the 165-kDa derivative. These results provide experimental clues for the interaction of S1 with actin subdomain 2 and show that this contact is implicated in the weak acto-S1 binding state. Furthermore they demonstrate the ability of Tm to affect the structure of actin subdomain 2 even in the presence of S1 bound in the rigor state.

CC Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Minerals 10069
Biophysics - Molecular properties and macromolecules 10506
Enzymes - Physiological studies 10808
Muscle - Physiology and biochemistry 17504
In vitro cellular and subcellular studies 32600

IT Major Concepts
Biochemistry and Molecular Biophysics; Enzymology (Biochemistry and Molecular Biophysics); Muscular System (Movement and Support)

IT Chemicals & Biochemicals
GLUTARALDEHYDE; INACTIN; CALCIUM

IT Miscellaneous Descriptors
CALCIUM; TROPONIN

ORGN Classifier
Leporidae 86040
Super Taxa
Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
rabbit
Taxa Notes

ANSWER 3 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 1994:212708 BIOSIS
DN PREV199497225708
TI Tropomyosin inhibits the glutaraldehyde-induced cross-link between the central 48-kDa fragment of myosin head and segment 48-67 inactin subdomain 2.
AU Bonafe, Nathalie; Mathieu, Magali; Kassab, Ridha; Chaussepied, Patrick [Reprint author]
CS Centre de Recherches de Biochemie Macromoleculaire, Route de Mende, BP 5051, 34033 Montpellier Cedex, France
SO Biochemistry, (1994) Vol. 33, No. 9, pp. 2594-2603.
CODEN: BICHAW. ISSN: 0006-2960.
DT Article
LA English
ED Entered STN: 10 May 1994
Last Updated on STN: 25 Jun 1994
AB The glutaraldehyde-induced cross-linking of the F-actin-myosin head (S1) complex, previously described (Bertrand et al. (1988) Biochemistry 27, 5728-5736), was investigated in the presence of tropomyosin (Tm) alone or associated with troponin (Tn), at a Tm-Tn/actin/S1 molar ratio of 1:7:3. Among the two acto-S1 cross-linked products with apparent masses of 165 and 200 kDa generated in the absence of the regulatory proteins, only the 165-kDa adduct was formed in the presence of Tm. An identical result was obtained with and without Tn regardless of the presence of Ca-2+ and/or Mg-2+-ADP. The abolition of the 200-kDa cross-linked acto-S1 species was independent of the S1/actin ratio since even a 3-fold excess of S1 over actin, sufficient for fully turning on the thin filament, could not restore the 200-kDa covalent involved in the Ca-2+-linked regulation of the acto-S1 ATPase activity, as the enzymatic activities of both types of complexes were regulated to the same extent by Ca-2+/EGTA, in the presence of the regulatory proteins. Cross-linking experiments performed with (14C) glutaraldehyde showed that both covalent complexes were composed of 1 mol of actin bound to 1 mol of S1 heavy chain. The use of proteolytic actin or S1 derivatives together with the direct proteolysis of the acto-S1 covalent adducts revealed that Tm abolished the cross-link between the central 48-kDa fragment of the S1 heavy chain and Lys-50 of actin subdomain 2 that is responsible for the formation of the 200-kDa entity, while it did not affect the cross-link between the S1 heavy chain segment of residues 636-642 and Arg-28 of actin that generates the 165-kDa derivative. These results provide experimental clues for the interaction of S1 with actin subdomain 2 and show that this contact is implicated in the weak acto-S1 binding state. Furthermore they demonstrate the ability of Tm to affect the structure of actin subdomain 2 even in the presence of S1 bound in the rigor state.
CC Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Minerals 10069
Biophysics - Molecular properties and macromolecules 10506
Enzymes - Physiological studies 10808
Muscle - Physiology and biochemistry 17504
In vitro cellular and subcellular studies 32600
IT Major Concepts
Biochemistry and Molecular Biophysics; Enzymology (Biochemistry and Molecular Biophysics); Muscular System (Movement and Support)
IT Chemicals & Biochemicals
GLUTARALDEHYDE; INACTIN; CALCIUM
IT Miscellaneous Descriptors
CALCIUM; TROPONIN
ORGN Classifier
Leporidae 86040
Super Taxa
Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
rabbit
Taxa Notes

Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates

RN 111-30-8 (GLUTARALDEHYDE)
947-08-0 (INACTIN)
7440-70-2 (CALCIUM)

Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates

RN 111-30-8 (GLUTARALDEHYDE)
947-08-0 (INACTIN)
7440-70-2 (CALCIUM)

ANSWER 8 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
AN 1995:224483 CAPLUS
DN 122:3074
ED Entered STN: 04 Dec 1994
TI Oxygen radicals attenuate the contractility of skinned muscle fibers from the pig myocardium
AU Loewe, H.; Baeger, I.; Blasig, I. E.; Haseloff, R. F.
CS Forschungsinstitut fuer Molekular Pharmakologie, Forschungsverbund e. V., Berlin, Germany
SO Pharmazie (1994), 49(11), 845-9
CODEN: PHARAT; ISSN: 0031-7144
PB Govi-Verlag Pharmazeutischer Verlag
DT Journal
LA English
CC 4-3 (Toxicology)
AB The radicals were generated by xanthine-xanthine oxidase (X/XO) or by Fe2+/H2O2 (Fenton system). Addition of the X/XO to the incubation medium (KCl/imidazole) induced a depression of the contractility which was dependent from the incubation time and the X/XO concentration The maximum contraction in the presence of high concns. of free calcium ions (pCa 4.32) decreased to 52.0 ± 15.5% ($p < 0.01$). The EC50 of calcium ions inducing fiber contraction increased from 2.28 ± 0.66 μmol/L to 5.47 ± 2.06 μmol/L ($p < 0.05$). The Hill plot of contraction vs. concentration of calcium ions was shifted to the right and the maximum of contractility was attenuated. Replacement of X/XO by the Fenton system was without significant effects on the fiber contractility. Addition of 5·10-4 mol/L APP 210-533 (3-amino-6-methyl-5-phenyl-1,2-dihydropyrid-2-one), a known "calcium sensitizer", increased the fiber contractility in radical impaired fibers, too. This may indicate that the radicals did not impair the troponine complex. Oxygen radicals were detected by ESR spectroscopy spin trapping using 5,5-dimethylpyrroline 1-oxide. Superoxide radicals were found in the presence of X/XO whereas addition of Fe2+/H2O2 to the incubation medium resulted in the formation of hydroxyl radical adducts. The appearance of addnl. adducts observed in both system is discussed. The expts. indicate that free radicals can interact with components of the skinned fiber (probably with contractile proteins of the myocardial muscle cells) resulting in an impairment of the contractility. This could be partly responsible for the "stunning phenomenon" in ischemic/reperfused myocardium, a reversible but long lasting deterioration of the myocardial contractility. The treatment of stunned myocardium with "calcium sensitizers" could be beneficial for the myocardial function.
ST oxygen radical heart contractility
IT Heart
(oxygen radicals attenuate the contractility of skinned muscle fibers from myocardium)
IT Reactive oxygen species
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(oxygen radicals attenuate the contractility of skinned muscle fibers from myocardium)
IT 3352-57-6, Hydroxyl, biological studies 7782-44-7D, Oxygen, radicals 11062-77-4, Superoxide
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(oxygen radicals attenuate the contractility of skinned muscle fibers from myocardium)
IT 7440-70-2, Calcium, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(oxygen radicals attenuate the contractility of skinned muscle fibers from myocardium as function of calcium)

ANSWER 8 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN

AN 1995:224483 CAPLUS

DN 122:3074

ED Entered STN: 04 Dec 1994

TI Oxygen radicals attenuate the contractility of skinned muscle fibers from the pig myocardium

AU Loewe, H.; Baeger, I.; Blasig, I. E.; Haseloff, R. F.

CS Forschungsinstitut fuer Molekular Pharmakologie, Forschungsverbund e. V., Berlin, Germany

SO Pharmazie (1994), 49(11), 845-9
CODEN: PHARAT; ISSN: 0031-7144

PB Govi-Verlag Pharmazeutischer Verlag

DT Journal

LA English

CC 4-3 (Toxicology)

AB The radicals were generated by xanthine-xanthine oxidase (X/XO) or by Fe2+/H2O2 (Fenton system). Addition of the X/XO to the incubation medium (KCl/imidazole) induced a depression of the contractility which was dependent from the incubation time and the X/XO concentration The maximum contraction in the presence of high concns. of free calcium ions (pCa 4.32) decreased to 52.0 ± 15.5% ($p < 0.01$). The EC50 of calcium ions inducing fiber contraction increased from 2.28 ± 0.66 μmol/L to 5.47 ± 2.06 μmol/L ($p < 0.05$). The Hill plot of contraction vs. concentration of calcium ions was shifted to the right and the maximum of contractility was attenuated. Replacement of X/XO by the Fenton system was without significant effects on the fiber contractility. Addition of 5·10-4 mol/L APP 210-533 (3-amino-6-methyl-5-phenyl-1,2-dihydropyrid-2-one), a known "calcium sensitizer", increased the fiber contractility in radical impaired fibers, too. This may indicate that the radicals did not impair the troponine complex. Oxygen radicals were detected by ESR spectroscopy spin trapping using 5,5-dimethylpyrroline 1-oxide. Superoxide radicals were found in the presence of X/XO whereas addition of Fe2+/H2O2 to the incubation medium resulted in the formation of hydroxyl radical adducts. The appearance of addnl. adducts observed in both system is discussed. The expts. indicate that free radicals can interact with components of the skinned fiber (probably with contractile proteins of the myocardial muscle cells) resulting in an impairment of the contractility. This could be partly responsible for the "stunning phenomenon" in ischemic/reperfused myocardium, a reversible but long lasting deterioration of the myocardial contractility. The treatment of stunned myocardium with "calcium sensitizers" could be beneficial for the myocardial function.

ST oxygen radical heart contractility

IT Heart
(oxygen radicals attenuate the contractility of skinned muscle fibers from myocardium)

IT Reactive oxygen species
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(oxygen radicals attenuate the contractility of skinned muscle fibers from myocardium)

IT 3352-57-6, Hydroxyl, biological studies 7782-44-7D, Oxygen, radicals 11062-77-4, Superoxide
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(oxygen radicals attenuate the contractility of skinned muscle fibers from myocardium)

IT 7440-70-2, Calcium, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(oxygen radicals attenuate the contractility of skinned muscle fibers from myocardium as function of calcium)

ANSWER 1 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 1996:109721 BIOSIS
DN PREV199698681856
TI Covalent binding of peptides to the N-terminal hydrophobic region of cardiac troponin C has limited effects on function.
AU Lin, Xin; Dotson, Darrell G.; Putkey, John A. [Reprint author]
CS Dep. Biochem. Mol. Biol., Univ. Tex. Med. Sch., 6431 Fannin St., Houston, TX 77030, USA
SO Journal of Biological Chemistry, (1996) Vol. 271, No. 1, pp. 244-249.
CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 12 Mar 1996
Last Updated on STN: 13 Mar 1996
AB Exposure of an N-terminal hydrophobic region in troponin C is thought to be important for the regulation of contraction in striated muscle. To test this hypothesis, single Cys residues were engineered at positions 45, 81, 84, or 85 in the N-terminal hydrophobic region of cardiac troponin C (cTnC) to provide specific sites for attachment of blocking groups. A synthetic peptide, AcVal-Arg-Ala-Ile-Gly-Lys-Leu-Ser-Ser, or biotin was coupled to these Cys residues, and the covalent adducts were tested for activity in TnC-extracted myofibrils. Covalent modification of cTnC(C45) had no effect on maximal myofibril ATPase activity. Greatly decreased myofibril ATPase activity (70-80% inhibited) resulted when the peptide was conjugated to Cys-81 in cTnC(C81), while a lesser degree of inhibition (10-25% inhibited) resulted from covalent modification of cTnC(C84) and cTnC(C85). Inhibition was not due to an altered affinity of the cTnC(C81)/peptide conjugate for the myofibrils, and the Ca-2+ dependence of ATPase activity was essentially identical to the unmodified protein. Thus, a subregion of the N-terminal hydrophobic region in cTnC is sensitive to disruption, while other regions are less important or can adapt to rather bulky blocking groups. The data suggest that Ca-2+--sensitizing drugs may bind to the N-terminal hydrophobic region on cTnC but not interfere with transmission of the Ca-2+ signal.
CC Cytology - Animal 02506
Biochemistry studies - Nucleic acids, purines and pyrimidines 10062
Biochemistry studies - Proteins, peptides and amino acids 10064
Biophysics - Molecular properties and macromolecules 10506
Enzymes - Chemical and physical 10806
Cardiovascular system - Physiology and biochemistry 14504
Muscle - Physiology and biochemistry 17504
IT Major Concepts
Biochemistry and Molecular Biophysics; Cardiovascular System (Transport and Circulation); Cell Biology; Enzymology (Biochemistry and Molecular Biophysics); Muscular System (Movement and Support)
IT Chemicals & Biochemicals
CALCIUM ION; ATPASE
IT Miscellaneous Descriptors
ATPASE ACTIVITY; CALCIUM ION SIGNAL TRANSMISSION; STRIATED MUSCLE
ORGN Classifier
Animalia 33000
Super Taxa
Animalia
Organism Name
Animalia
Taxa Notes
Animals
RN 14127-61-8 (CALCIUM ION)
9000-83-3 (ATPASE)

ANSWER 1 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 1996:109721 BIOSIS
DN PREV199698681856
TI Covalent binding of peptides to the N-terminal hydrophobic region of cardiac troponin C has limited effects on function.
AU Lin, Xin; Dotson, Darrell G.; Putkey, John A. [Reprint author]
CS Dep. Biochem. Mol. Biol., Univ. Tex. Med. Sch., 6431 Fannin St., Houston, TX 77030, USA
SO Journal of Biological Chemistry, (1996) Vol. 271, No. 1, pp. 244-249.
CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 12 Mar 1996
Last Updated on STN: 13 Mar 1996
AB Exposure of an N-terminal hydrophobic region in troponin C is thought to be important for the regulation of contraction in striated muscle. To test this hypothesis, single Cys residues were engineered at positions 45, 81, 84, or 85 in the N-terminal hydrophobic region of cardiac troponin C (cTnC) to provide specific sites for attachment of blocking groups. A synthetic peptide, AcVal-Arg-Ala-Ile-Gly-Lys-Leu-Ser-Ser, or biotin was coupled to these Cys residues, and the covalent adducts were tested for activity in TnC-extracted myofibrils. Covalent modification of cTnC(C45) had no effect on maximal myofibril ATPase activity. Greatly decreased myofibril ATPase activity (70-80% inhibited) resulted when the peptide was conjugated to Cys-81 in cTnC(C81), while a lesser degree of inhibition (10-25% inhibited) resulted from covalent modification of cTnC(C84) and cTnC(C85). Inhibition was not due to an altered affinity of the cTnC(C81)/peptide conjugate for the myofibrils, and the Ca-2+ dependence of ATPase activity was essentially identical to the unmodified protein. Thus, a subregion of the N-terminal hydrophobic region in cTnC is sensitive to disruption, while other regions are less important or can adapt to rather bulky blocking groups. The data suggest that Ca-2+--sensitizing drugs may bind to the N-terminal hydrophobic region on cTnC but not interfere with transmission of the Ca-2+ signal.
CC Cytology - Animal 02506
Biochemistry studies - Nucleic acids, purines and pyrimidines 10062
Biochemistry studies - Proteins, peptides and amino acids 10064
Biophysics - Molecular properties and macromolecules 10506
Enzymes - Chemical and physical 10806
Cardiovascular system - Physiology and biochemistry 14504
Muscle - Physiology and biochemistry 17504
IT Major Concepts
Biochemistry and Molecular Biophysics; Cardiovascular System (Transport and Circulation); Cell Biology; Enzymology (Biochemistry and Molecular Biophysics); Muscular System (Movement and Support)
IT Chemicals & Biochemicals
CALCIUM ION; ATPASE
IT Miscellaneous Descriptors
ATPASE ACTIVITY; CALCIUM ION SIGNAL TRANSMISSION; STRIATED MUSCLE
ORGN Classifier
Animalia 33000
Super Taxa
Animalia
Organism Name
Animalia
Taxa Notes
Animals
RN 14127-61-8 (CALCIUM ION)
9000-83-3 (ATPASE)

ORGN Classifier
Leporidae 86040
Super Taxa
Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
rabbit
Taxa Notes
Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates
RN 60267-61-0 (UBIQUITIN)
9031-56-5 (LIGASE)
9080-13-1Q (LIGASE)

ORGN Classifier
Leporidae 86040
Super Taxa
Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
rabbit
Taxa Notes
Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates
RN 60267-61-0 (UBIQUITIN)
9031-56-5 (LIGASE)
9080-13-1Q (LIGASE)